lowing percutaneous transluminal septal ablation for HCM. The data also support prior assertions that much of the CRP response observed in MI is driven by the infarcted myocardium, rather than the atherosclerotic plaque.

MODERATED POSTER SESSION

1169MP Moderated Poster Session...Basic Correlates of Myocardial Ischemia/ Reperfusion

Tuesday, March 19, 2002, Noon-2:00 p.m.
Georgia World Congress Center, Hall G

Noon

1169MP-121 Correlation of Heat Production of Culprit Atherosclerotic Lesion With Soluble Cell Adhesion Molecules

Konstantinos Toutouzas, Christodoulos Stefanadis, Eleftherios Tsiamis, Manolis Vavouranakis, Ioannis Kallikazaros, Sophia Vaina, Christina Chrysochoou, Dimos Panagiotakos, Marina Toutouza, Pavlos Toutouzas, *Hippokration Hospital, Athens, Greece.*

Cell adhesion molecules are critical markers of the inflammatory process, which is involved in the pathogenesis of coronary artery disease (CAD). Previous ex vivo and in vivo studies have shown thermal heterogeneity within human atherosclerotic plaques. The purpose of the present study was to measure the luminal surface temperature in patients with CAD and to correlate it with the soluble cell adhesion molecules in order to evaluate the role of inflammation in heat production in acute coronary syndromes. **Methods**: In the study we included 25 patients (pts) with CAD [(12 with myocardial infarction (MI) during the last month and 13 with unstable angina (UA)] and 10 sex- and age-matched controls without CAD. In all pts plasma levels of soluble inter-cellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule (VCAM-1) were measured. A thermography catheter developed in our Institution was used, in order to measure intracoronary temperature. A thermistor probe with a temperature accuracy of 0.05 °C, was attached at the distal end of a long 3F polyurethane shaft. Thus, we measured the median temperature differences at the site of the lesion from the core temperature (TD). **Results:** The median temperature differences at the site of the lesion from the core temperature (TD) were increased in patients with MI (0.59 ± 0.19 °C) and UA (0.27 ± 0.16 °C) ($p<0.001$). Levels of VCAM-1 and ICAM-1 concentrations were increased in pts with CAD compared with the control group (VCAM-1: 571.2 ± 169.7 ng/ml vs. 406.1 ± 131.2 ng/ml, $p < 0.05$; ICAM-1: 343.6 ± 107.3 ng/ml vs. 274.5 ± 73 .8 ng/ml, $p < 0.05$). Additionally, a good correlation was observed between levels of VCAM-1 with TD ($r=0.53$, $P=0.01$). Also, a correlation with ICAM-1 was also observed without however reaching statistical significance. **Conclusion**: An aggressive inflammatory response occurring in acute coronary syndromes results in increased local heat production. This suggests that, temperature measurement of culprit lesions may be used in future studies to evaluate the effect of anti-inflammatory regimens on the atherosclerotic plaque stabilization.

12:12 p.m.

1169MP-122 Endogenous Endothelin-1 Reduces the Postischemic Functional Recovery of Prolonged Hypoperfused Myocardium via the Endothelin-A Receptor

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Background: The release of endothelin-1 (ET-1) from the damaged endothelium may play a role in the initiation and maintenance of myocardial ischemia. This study examines the ETA-receptor mediated role of endogenous endothelin on postischemic myocardial function after prolonged hypoperfusion.
Methods: In an isolated rat heart model for short-term hibernation the left ventricular functional recovery after 3h hypoperfusion (15% of preischemic coronary flow) followed by 2h reperfusion was determined (isovolumic steady state hemodynamics: coronary flow, left ventricular pressure (LVP), dP/dtmax; maximal inotropic response to calcium stimulation: max LVP). The effect of ET conversion inhibition by phosphoramidon (PHOSPH, 2 µmol/l) and of ETA-blockade by BQ 610 (0.8 µmol/l) during hypoperfusion was compared to saline controls (NaCl).
Results: 2h of reperfusion after hypoperfusion causes a partial functional recovery. This postischemic functional recovery is significant better with ECE-inhibition or with ETA-blockade during hypoperfusion. (The table shows mean ± SEM of preischemic values after 2h of reperfusion; *=p<0.05, §=p<0.01, #=p<0.001 versus NaCl-group.)
Conclusion: Reperfused myocardium profits from ECE-inhibition or ETA-blockade during the prolonged period of hypoperfusion indicating that its postischemic functional recovery is reduced by endogenous ET-1 via ETA-receptors.

	coronary flow	LVP	dP/dtmax	max LVP	max dP/ dtmax
PHOSPH	57.7±4.1	58.2±2.5#	61.9±3.7#	73.3±10.3	74.1±17.0
BQ 610	54.1±5.0	56.0±2.5#	54.8±2.8§	85.1±5.6*	84.5±6.3
NaCl	49.3±2.4	36.5±2.1	41.1±1.1	58.6±5.6	59.1±13.5

12:24 p.m.

1169MP-123 Vasomotor Function of Pig Coronary Arteries After Placement of Ameroid Constrictors

Jin-Shen Li, Takafumi Ueno, Hector de Leon, Jianhua Cui, Patrick K. Coussement, Nicolas Chronos, Keith A. Robinson, *Atlanta Cardiovascular Research Institute, Norcross, Georgia.*

Background: Placement of ameroid constrictors in coronary arteries of pigs causes progressive stenosis and distal myocardial ischemia. Blood perfusion in the ischemic region is partly dependent on vasomotor responses to neural and humoral factors, in the stenotic or occluded artery. We studied vascular function in epicardial arteries distal to the site of ameroid constrictors at 8 wk after placement, to assess whether their responses to endothelium-dependent and -independent vasorelaxants as well as vasoconstrictors, differed from adjacent normal epicardial arteries. **Methods:** Four-mm rings of LCX distal to constrictor placement (n=12) and identical size LAD (n=8) were obtained from 6 juvenile crossbred pigs 8 wk after ameroid placement and endothelium-dependent or -independent functions were studied in an organ chamber system. **Results:** Contractions to 40mM & 100mM KCl were similar, but contraction to 30 mM PGF2a was lower in LCX than LAD (4.63±0.28g in LCX & 5.09±0.37g in LAD). After nitric oxide synthase inhibition using L-NAME, contraction to 30 mM PGF2a was increased in LCX (4.63±0.28 g vs. 6.25±0.30 g, $P<0.001$). Endothelium-dependent relaxation to 100 pM substance P was nearly abolished by L-NAME in LCX (60.5±6.2% vs. 13.6±3.0%, $P<0.001$). Reduction of the endothelium-dependent relaxation was significantly greater in the LCX than in the LAD (77.0±0.04% vs. 59.0±0.03%, $P<0.01$). Endothelium-independent relaxation to 100 nM sodium nitroprusside (SNP) was similar in LCX and LAD, however, both arteries were significantly more sensitive to the same dose of SNP after NO blockade with L-NAME (32.6±5.3% to 76.7±2.8% in LCX, $P<0.001$ & 25.4±5.4% to 71.3±2.9% in LAD, $P<0.001$). **Conclusions:** The pig coronary artery showed adaptive responses 8 wk after ameroid constrictor placement, which would tend to abrogate myocardial ischemia via decreasing vascular tone. This adaptation may in part involve changes in nitric oxide pathways since the decreased contraction and increased relaxation responses of the affected coronary arteries were partially inhibited by L-NAME.

12:36 p.m.

1169MP-124 Heterogeneous Perfusion Insufficiency and Three-Dimensional Microstructure Abnormality of Coronary Capillary Network After Myocardial Reperfusion

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Background: Coronary perfusion insufficiency is known to occur heterogeneously at the capillary network level depending on the minimum size of coronary flow control unit (FCU; several hundreds µg length). We aimed to investigate micro-perfusion pattern and three-dimensional [3-D] structural abnormality of coronary capillary network after myocardial reperfusion using a confocal laser scanning microscopy (CLSM).
Methods: Using opened-chest anesthetized Wistar rats' hearts, LAD was occluded for 7 min followed by 3 min reperfusion. The hearts were divided into two groups; 1) well stained reperfused area by indocyanine green iv after reperfusion [Good-reflow], and 2) poorly stained group [No-reflow]. Then, the hearts were isolated and perfused by Langendorff's mode. Entire coronary microvasculature was filled with contrast medium [BaSO4+Indian ink+gelatin]. Capillaries in the section [200µm in thickness] were observed 3-dimensionaly using CLSM [0.09µm3/voxel] for control area [Ct; LCX region] and reperfused area [LAD region] in both reflow groups. Capillary volume fraction [CVF=capillary vol./[myocardial vol.+ capillary vol.] was computed from 3-D images.
Results: Comparing with Ct, reperfused area of both groups showed decreased capillary diameter and density with waving and shrinkage configuration. CVF was significantly reduced by 40% in the reperfused area of Good-reflow compared with Ct [p<.005], and further decreased by 83% in No-reflow [P<.001, vs. Ct, p<.001, vs. Good-reflow]. In No-reflow, the low perfusion area was distributed heterogeneously with similar low-flow clusterings of several FCUs lower flows.
Conclusions: Coronary no-reflow after myocardial reperfusion was characterized by heterogeneous capillary filling reduction with morphological change such as waving and shrinkage.

12:48 p.m.

1169MP-125 Ischemic Preconditioning Protects the Heart From Membrane Current Changes Due to Ischemia

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Background: The use of ischemic preconditioning (IPC) has been suggested to protect hearts undergoing surgically induced ischemia. Although it is known that IPC protects against infarction, the effect of IPC on the transport of ions via sarcolemmal channels is unknown. We hypothesize that IPC protects against changes in K^+ membrane current after ischemia. **Methods:** Isolated rabbit hearts were mounted on a Langendorff apparatus and perfused for 20 min. Control hearts were not exposed to ischemia, the ischemic group was exposed to 60 min of ischemia, and the IPC group was exposed to 2 episodes of 5-min of ischemia/reperfusion prior to 60 min of ischemia. Ventricular myocytes were then isolated. Using the whole cell patch clamp technique, the current-voltage (I-V) relationships were determined. Ba^{2+} was applied to block inwardly rectifying K^+ current. **Results:** Ischemia resulted in a significant change in the Ba^{2+} sensitive portion of the